

# Dual threat of comorbidity of celiac disease and systemic lupus erythematosus

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## Abstract

Celiac disease (CD) is a chronic immune-mediated intestinal disease that is characterized by production of autoantibodies directed against the small intestine. The main clinical manifestations of CD are typically defined as those related to indigestion and malabsorption. These manifestations include unexplained diarrhea or constipation, abdominal pain, bloating, weight loss, anemia, failure-to-thrive in children, and decreased bone density. Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by heterogeneous clinical manifestations, which may also involve the gastrointestinal tract. Comorbidity of CD and SLE is rare, and the overlapping symptoms and nonspecific clinical presentation may pose a diagnostic challenge to clinicians. We report here a case of SLE with CD, which mainly manifested as recurrent diarrhea, uncorrectable electrolyte disorders, and severe malnutrition. Through review, we hope to further improve our understanding and diagnostic level of this combination of diseases.

## Keywords

Celiac disease, systemic lupus erythematosus, comorbidity, gastritis, diarrhea, weight loss

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## Introduction

Celiac disease (CD) frequently has gastrointestinal and extraintestinal manifestations, and it is triggered by ingestion of gluten in genetically predisposed individuals.<sup>1</sup> CD has an incidence of approximately 1% in the general population, and it frequently occurs in Western Europe, but is

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rare in Asia.<sup>2</sup> Especially in China, large-sample epidemiological data of CD are currently lacking. Typical symptoms of CD include diarrhea, indigestion, weight loss, and signs of malabsorption. However, some patients with CD show extraintestinal symptoms, such as dermatitis herpetiformis, thyroid dysfunction, autoimmune hepatitis, coagulation dysfunction, or even neurological symptoms.<sup>3</sup> Systemic lupus erythematosus (SLE) is an autoimmune disease that can involve multiple organs. The coexistence of CD and SLE has rarely been reported.<sup>4</sup> We describe here a case of comorbidity of these two diseases, and highlight shortcomings of the current understanding and emphasize the requirement for improving their diagnostic level.

## Case report

A 54-year-old woman with a 4-month history of diarrhea, mild lower abdominal pain, fatigue, anorexia, painful muscle cramps, and considerable weight loss visited our clinic because the discomfort had worsened over the past month. She had a 10-year history of SLE and was treated with oral prednisone intermittently. Because her symptom of joint pain was well controlled, she had decided to stop taking prednisone for nearly 1 month. Her hair began to fall out 8 years previously. She had no history of smoking or drinking alcohol, and no family history of colorectal cancer or inflammatory bowel disease.

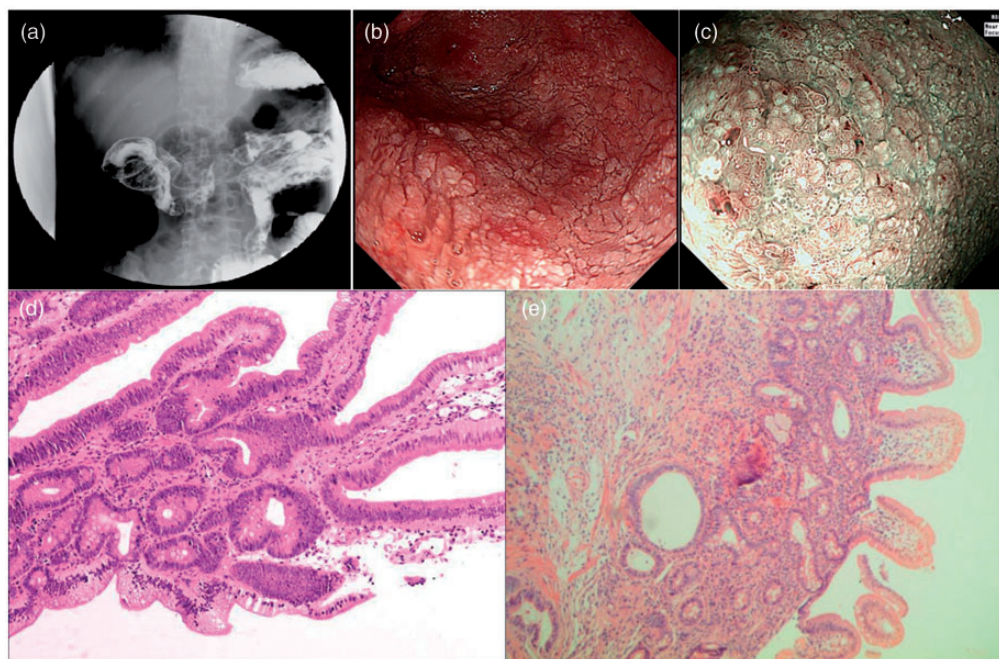
The body mass index of this patient was 11.9 kg/m<sup>2</sup> and an abdominal physical examination was unremarkable. Laboratory studies showed the following: hemoglobin level, 98 g/L (normal, 110–150 g/L); platelet count,  $77 \times 10^9$ /L ( $100\text{--}300 \times 10^9$ /L); albumin level, 22.6 g/L (35–50 g/L); serum potassium level, 3.01 mmol/L (3.5–5.5 mmol/L); erythrocyte sedimentation rate, 66 mm/hour (0–20 mm/hour); immunoglobulin A level, 6.93 g/L (7.6–39 g/L); immunoglobulin G level,

23.1 g/L (7.0–17.0 g/L); immunoglobulin E level, 374 IU/mL (0–165.3 IU/mL); complement component C3 level, 0.47 g/L (0.8–1.2 g/L); anti-nuclear antibodies (+); anti-nuclear ribonucleoprotein/anti-Smith antibodies (+); anti-Sjogren's syndrome antigen A antibodies (+); anti-Ro-52 antibodies (+); anti-mutated citrullinated vimentin antibodies, 42.8 U/mL (0–20 U/mL); and 24-hour urine protein quantification, 354 mg (<150 mg). Digestive tract radiography suggested segmental stenoses of the duodenum (Figure 1a). Gastroscopy showed gastritis and duodenal mucosal atrophy with cobblestoning (Figure 1b, c). Colonoscopy showed no obvious abnormality. Histology of the duodenal biopsies was consistent with CD, and was characterized by total villous atrophy, crypt hyperplasia (Figure 1d), and an increased number of intraepithelial lymphocytes (Marsh IIIC) (Figure 1e). After treatments with albumin supplementation, electrolyte correction, and enteral and parenteral nutritional support, the patient's symptoms greatly improved. Reporting of this study conforms to the CARE guidelines.<sup>5</sup>

## Discussion

CD is an immune-mediated disorder of the gastrointestinal tract and mainly manifests as immune destruction of the inner wall of the small intestine and the villous atrophy.<sup>6</sup> Insufficient understanding of this disease in the clinical situation often delays its diagnosis and affects the prognosis of patients.

Serological antibody detection is an important method of screening and diagnosing CD. This approach contains endomysial antibody, anti-tissue transglutaminase antibody, deamidated antigliadin antibody, and anti-gliadin antibody. A prospective cohort study showed that anti-tissue transglutaminase antibody and endomysial antibody testing had a higher sensitivity and specificity than anti-gliadin antibody testing.<sup>7</sup> Deamidated anti-gliadin antibody is a new



**Figure 1.** Digestive tract radiography suggests segmental stenoses of the duodenum (a). Gastroscopy shows gastritis and duodenal mucosal atrophy with cobblestoning (b, c). Histology of the duodenal biopsies is consistent with celiac disease, which is characterized by total villous atrophy, crypt hyperplasia (d), and an increased number of intraepithelial lymphocytes (Marsh IIIc) (e).

antibody marker and plays a complementary role to the other related antibodies. Therefore, this antibody has become increasingly used in the diagnosis of CD in recent years.<sup>8</sup> Histopathology of the duodenum is the most common method for definitive diagnosis of CD. A guideline states that one or two biopsies of the duodenal bulb and at least four biopsies of the post-bulbar duodenum could improve the positive rate of CD.<sup>9</sup> The typical histopathological features of CD include partial or total villous atrophy, crypt hyperplasia, and an increased number of intraepithelial lymphocytes (>25/100 enterocytes).<sup>10</sup> In addition, genetic factors, such as human leukocyte antigen (HLA)-DQ2 and HLA-DQ8, are considered to play an important role in the pathogenesis of CD. Previous studies have suggested that approximately 90% of

patients with CD carry the HLA-DQ2 gene, while 5% carry the HLA-DQ8 gene. Detection of the HLA-DQ2/DQ8 genotype has a high sensitivity for the diagnosis of CD.<sup>11</sup>

SLE is a chronic autoimmune disease with damage to multiple systems. The etiology of SLE is still unclear. Abnormal activation of the human immune system is thought to attack the tissue, resulting in organ damage and a series of clinical symptoms. There are some similarities between SLE and CD in the course of onset. SLE can involve the gastrointestinal tract and overlaps with CD in clinical symptoms. All of these factors result in a diagnostic challenge to clinicians.

In our case, the diagnosis of SLE was much earlier than CD. However, we could not determine the real onset time of CD

owing to the lack of understanding of this disease and the limitations of detection methods. In addition, this patient had taken malt extract for approximately 1.5 months before the onset of CD, which may have triggered sudden aggravation of CD. CD manifested as diarrhea and severe weight loss, anemia, and electrolyte disturbance. A gluten-free diet is the basis of treatment for CD.

### Ethics statement

The patient provided written informed consent for publication of this report. We de-identified all of the patient's details and obtained consent for treatment. This was a retrospective case report; therefore, institutional review board approval was not required.

### Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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